Kee et al (1996), (abstract) states: "Individuals with DPT demonstrated significantly reduced forced vital capacity (FVC) (p=0.002) and diffusing capacity for carbon monoxide (DLCO) (p=0.002) as compared with the referents.

Lee et al (2003), p.205, states with regard to 38 subjects with Wittenoom crocidolite exposure: "In our study, pleural thickening was associated with reduced lung volumes and DLCO (as had been shown by others 34, 35)."

Yates (1996), p.304, states with regard to 64 patients with diffuse pleural thickening: "Our study confirms the findings of earlier workers in that a significant decrement of FEV_1 and FVC was observed (3-12, 15-22)."

Kouris et al (1991) studied workers manufacturing wallboard and ceiling tile, exposed to predominately chrysotile asbestos. 22 subjects had diffuse pleural thickening. The authors write that "Diffuse thickening was associated with more profound decreases in FVC and FEV1.0."

Copley et al (2001), p.240, states regarding a study of 50 patients in England: "Reductions in residual volume were linked to severity of diffuse pleural thickening."

ATS (2004) Official Statement, p.708, finds a decrease in "FEV1/FVC ratio also occurs in subjects who do not have radiographic evidence of

asbestosis (defined as an ILO score exceeding 1/0)," probably indicating a reduction due to pleural disease.

30. It is common knowledge that there is little correlation between radiographs and asbestos pleural disease severity, and certainly none that could be applied to individual patients. Miles et al (2008) "Clinical Consequences of Asbestos-related Diffuse Pleural Thickening, a Review," p.6, states: "Few longitudinal studies exist, but these have found no correlation between radiographic severity and longitudinal loss of lung function." Yates, et al (1996), "Asbestos re Bilateral Diffuse Pleural Thickening: Natural History of Radiographic and Lung Function Abnormalities," p.305, states: "No correlation was observed between radiographic severity and longitudinal loss of lung function. The chest radiograph even when accompanied by oblique films was an insensitive index of disease severity." Similarly, Whitehouse (2004), p.223, states: There was no statistical correlation between the extent of pleural changes measured on the chest x-ray and the loss of pulmonary function."

Similar statements have been presented in studies on the asbestos insulators. Markowitz et al (1997), p.106, states: "The chest x-ray can be normal even in the face of severe interstitial fibrosis." In the Markowitz et al (1997) study of 74 asbestosis deaths, 11% (8/74) had minimal or no

interstitial fibrosis (ILO score of 0/1 or less). Selikoff et al (1964), p.148, states: "significant disability may be present with relatively little to be seen on x-ray, and, conversely, x-ray changes may be extensive, with little functional difficulty."

E. CARD Mortality Study

31. A mortality study was done on patients of the Center for Asbestos Related Disease (CARD) Clinic in Libby. The cohort is all patients seen at CARD or by me in Spokane, with an asbestos-related disease diagnosis due to exposure to Libby asbestos and chest films. The study was done under my supervision.

Based upon CARD records, a total of 227 patients were identified as deceased through 7/9/08. There was no effort to track patients of uncertain status. 41 were excluded as having had no diagnosis of asbestos-related disease (ARD), no death certificate, no chart and/or no chest film, or as having had no exposure pre-1990. The total included in the mortality study for analysis is 186.

Data was gathered from patient charts. Available death certificates were obtained, including those available through the law firms of McGarvey, Heberling, Sullivan & McGarvey and Lewis, Slovak & Kovacich.

I reviewed patient charts and made determinations on whether

asbestos-related disease was a significant contributing factor in the death of each patient, using a method comparable to the "best available information" method described in Selikoff et al (1992), "Use of Death Certificates in Epidemiological Studies, Including Occupational Hazards: Variations in Discordance of Different Asbestos-Associated Diseases on Best Evidence Ascertainment." Since all subjects in the cohort were CARD patients, as treating physicians we often had important information in excess of that available to Selikoff et al. On the other hand, we did not have all hospital records on all the deceased. Where there was uncertainty, such that I could not determine that ARD was a significant contributing factor in the death to a medical probability, I did not call the death as an ARD death. Data is displayed on the chart "Summary of Mortality Disease Percentages," (Exh. 7). I determined that 110 of 186 (59%) deceased patients died of asbestos-related disease - 7 from mesothelioma, 19 from asbestos related lung cancer, 8 from other asbestos related cancer, and 76 from "asbestosis," which is defined as including asbestos related pleural disease per Markowitz et al (1997) Table 2. Note that non-malignant ARD deaths are generally coded to ICD-9 501 "Asbestosis." See Horton et al (2006) which publishes Libby ATSDR data. Table 1 "asbestosis" ICD-9 501 includes "known asbestos-related diseases."

Data are displayed on the spreadsheets listed at Exh. 7, a key to column headings the spreadsheets is as follows:

Column	Symbols
Exposure	W = Grace worker (employee) Sub = Subcontractor worker at Grace, not an employee FM = Family member of worker C = Community resident other than W or FM
SMK	S = current smoker N = never smoker Q = quit smoking
Pulm Hyp	Pulmonary hypertension
PFT - Dth months	Number of months, last PFT to death
Scales 0,1,2,3	0, 1 = minimal, 2 = moderate, 3 = extensive
Ext	Extent of chest wall

Other important observations include the following.

1) Only 34% (37/110) of those who died of non-malignant disease were mineworkers. 66% (73/110) were community members and family members of mineworkers. Exh. 7. Many community members had occupational exposures to Libby asbestos at the lumbermill and other jobs.

2) The death rate from asbestos disease was not greatly different as between mineworkers (69% - 37/54) who generally had heavy exposures, and community members (51% - 55/108), who had relatively light exposures.

3) Based upon the Dr. Frank chest x-ray readings, 26% (14/54) of

those who died of non-malignant disease died with pure pleural disease, with no interstitial fibrosis on the last chest x-ray. Of the 14, 7 had available CT scans. Of the 7, 2 had no interstitial disease on CT scan. Extending the above numbers to the 14, probably most of the 14 with no interstitial disease on chest x-ray did show some on CT scan. ATS (2004), p.707, reports only five total cases of death by pleural disease outside Libby.

4) Based upon the chest x-ray readings by Dr. Frank, all but one of 76 non-malignant ARD deaths had pleural disease to some degree. That one had significant interstitial fibrosis on CT scan. Two had no pleural disease on chest x-ray, but did have significant pleural thickening on CT scan. 26% (14/54) had no interstitial disease on chest x-ray. 56% (30/54) had minimal or no interstitial disease (0/1 or less), on chest x-ray. 55% (37/67) died with moderate or extensive pleural disease (on the spreadsheets moderate is a "2" and extensive is a "3"). 20 of the 37 (54%), had minimal or no interstitial fibrosis (ILO 0/1 or less). This means that many had moderate or extensive pleural disease, and minimal or no interstitial disease. The above confirms clinical observations that many patients die with extensive pleural disease and little or no interstitial disease. We find no similar reports of a high death rate by asbestos pleural disease elsewhere in the literature.

5) Based upon the Dr. Frank chest x-ray readings, 88% (61/69) had pleural thickening as defined by ATS (2004), Official Statement, p.707, ¶ 28 above. 12% (8/66) had pleural plaquing. Note that pleural plaquing often may not be seen where diffuse pleural thickening is extensive.

6) The last pulmonary function test on each non-malignant ARD death was reviewed. Using severity designations appropriate to Libby patients, (¶ 20 above), the following observations are made per Knudson norms for spirometry, Intermountain Thoracic Society for lung volumes and Miller for diffusion capacity. Use of Intermountain norms for spirometry and diffusion capacity will result in a significant decline in percentage predicted:

2 normal (FVC, TLC, DLCO all over 79)
7 mild (FVC, TLC, or DLCO between 70 and 79)
10 moderate (FVC, TLC, or DLCO between 60 and 69)
54 severe (FVC, TLC, or DLCO under 60)
3 no PFT
76 total

7) It is significant that 47% (29/61) had only DLCO under 65, not FVC or TLC. DLCO is a very important indicator of severity in the Libby cohort. For the 17 never smokers, the average DLCO on last PFT was 50% of predicted.

8) Of the non-malignant ARD deaths, 36% (26/72) had the FEV_1/FVC ratio under 65, indicating some obstructive defect.

9) Mean age at diagnosis was 69.47. Mean age at death was 76.1.

Based upon male life expectancy at diagnosis, an average 7.4 years of life expectancy was lost to asbestos disease. See Life Table cited at Exh. 5.

32. Comparison of CARD Mortality Study to Insulator Studies. The CARD mortality study found that in the group of patients diagnosed with ARD who have died, 43% died with ARD as a significant contributing factor per death certificate, and 59% died with ARD as a significant contributing factor per best available information analysis. Exhibit 7, Summary. The 59% death rate from asbestos disease appears to be the highest reported for any cohort in the United States.

The experience of the cohort of asbestos insulation workers provides a context for comparison of Libby mortality from asbestos related disease. The asbestos insulators had extremely heavy exposures to asbestos dust, often working in clouds of dust. The Libby patients, on the other hand, are mostly community members many of whom had relatively light exposure to asbestos from casual exposure, such as breathing the air in the Libby area. Only 34% (37/110) of the Libby patients who died of asbestos disease were mineworkers.

The cohort of asbestos insulation workers was first studied in Selikoff et al (1964). A number of follow-up studies have been performed, including Selikoff and Seidman (1991), "Asbestos Associated Deaths Among Insulation Workers in the U.S. and Canada, 1967-1987." The CARD mortality study data

have been adjusted for better comparability to the insulator studies, which used "primary cause" of death in best evidence determinations of cause of death, and underlying cause in death certificate analysis. See Exhibit 7a. Determinations of "primary cause" of death were done conservatively. If records were unclear or insufficient, primary cause was not found. The results of this mortality study and the CARD mortality study are compared in the following table.

Selikoff and Seidman (1991), p.7, Table 2 summary format; comparison to Libby CARD mortality study

	Insulators per DC		CARD per DC Exh. 7a		Insulators per BAI		CARD per BAI Exh. 7	
Lung cancer	1008	20%	16	9%	1168	24%	19	10%
pleural meso peritoneal meso	89 92	4%	7	4%	173 285	9%	7	4%
GI cancer	188	4%	6	3%	189	4%	8	4%
asbestosis	201	4%	39	21%	427	9%	76	34%
Total ARD deaths	1578	32%	68	37%	2242	45%	110	52%
All causes	4951	100%	186	100%	4951	100%	186	100%

DC - death certificate
BAI - best available information

One problem in comparing the two mortality studies is that in the CARD study all patients in the cohort were diagnosed with asbestos-related disease (ARD), whereas in Selikoff and Seidman (1991), there is no information on whether all the deceased had earlier been diagnosed with ARD. It can be estimated from Selikoff and Seidman (1991) that a very high percentage of the insulators who died likely had been diagnosed with ARD. From Table 4, we see that 96% (967/1,008) of the deaths from lung cancer were over 20 years from first exposure. From Table 6, we see that 96% (412/427) of the deaths from asbestosis were over 20 years from first exposure. From Selikoff et al (1964), Table 5, we can determine that 86% (339/392) of those workers over 20 years from first exposure had abnormal

chest x-rays. With the abnormal chest x-ray and exposure history, there is a likelihood of diagnosis of ARD. We conclude that the results displayed in the table above for the two studies are comparable. Even if we subtract 5% from the CARD death rate (since all CARD patients were diagnosed with ARD), the CARD death rate is still significantly higher than the insulators' death rate, despite the relatively light exposure of the CARD patients.

The Libby death rate from ARD is 52% per primary cause and best available information with 37% per underlying cause on death certificates. The insulators' death rate from ARD is 45% per best available information and 32% per death certificates. With a death rate higher than even the insulators cohort, it is apparent that exposure to Libby asbestos is considerably more toxic to humans than was the predominately chrysotile asbestos exposure of the insulation workers. (Regarding fiber type, see Seidman and Selikoff (1990), p.311).

Markowitz et al (1997), "Clinical Predictors of Mortality from Asbestosis in the North American Insulators Cohort, 1981 to 1991," followed 2,609 insulators examined in 1981 to 1983. The CARD mortality study may be compared to the Markowitz et al mortality study on asbestos insulators. The abstract states: "Seventy-four (11.0%) of 674 deaths during the subsequent 10 years were due to asbestosis, according to the best clinical and radiological evidence available at the time of death." The CARD mortality

study used similar methods. Results are compared as follows:

	Markowitz et al (1997)	CARD Mortality Study
Mean age at examination	57.5	69.5
Mean age at death	65.9	76.1
Years exam to death	8.4	6.6
Asbestosis deaths	74	62
Asbestosis deaths as % of total deaths	11%	34%
No pleural abnormalities on CXR	11%	1%
No interstitial abnormalities on CXR	4%	34%
Interstitial 0/1 or less on CXR	11%	36%
Interstitial 2/1 or more on CXR	55%	19%

Though the studies differ in minor respects, they are epidemiologically comparable. Markowitz et al (1997) followed individual insulators for 10 years in sequence. In the CARD study, patients were followed from diagnosis to death, a period averaging 6.6 years, but in some cases was over 10 years. These studies are nevertheless comparable. Both studies used the designation "asbestosis," despite varying degrees of pleural and interstitial disease.

The insulators had predominately interstitial disease, whereas the CARD patients had predominately pleural disease. The insulators were about 3x as likely to have severe interstitial disease (55% to 19%). The

CARD patients were about 3x as likely to have minimal interstitial disease (36% to 11%). Of the insulators group, 11% (8/74) were category zero for interstitial fibrosis. Of the eight in this group, seven had pleural thickening. Markowitz et al (1997), p.104. Of the CARD patients, 34% (24/71) were category zero for interstitial fibrosis on chest x-ray. All but one of the 24 had pleural disease on chest x-ray, and that one had both pleural and interstitial disease on CT scan.

Markowitz et al (1997) does not state the number who were diagnosed with asbestos-related disease on examination in 1981 to 1983. As discussed above, it is known that the incidence of asbestos-related disease was very high in the insulators with 30 years exposure. All patients in the CARD mortality study were diagnosed with asbestos-related disease.

The most striking observation is that the CARD patients' death rate from "asbestosis" is about 3x that of the insulators (34% to 11%). This result appears despite the important difference in exposure levels. 62% (47/76) of the CARD patients were family or community members with relatively light exposures (Exh. 7), whereas the insulators had industrial exposures to asbestos which were generally quite extreme. In the CARD mortality studies, persons who were not mineworkers or family members of mineworkers, were designated as "community members." Some had occupational exposures.

It is also observed that the CARD patients once diagnosed have a probability of death by asbestos-related disease, whereas the insulators do not. Markowitz et al (1997), Table 2, shows that no category (except ILO 3/4 which is exceedingly rare) of interstitial or pleural disease brings a probability of death.

F. Libby asbestos is highly toxic.

33. Many studies have been done on Libby asbestos and its toxicity. See the attached listing: "Libby Studies."

34. There generally appears to be a distinct pattern for Libby asbestos disease. The disease appears to be predominately pleural, for the large portion of the time that people have the disease. In the Whitehouse (2004) study, 55% of the patients followed had no interstitial disease and 45% had only minimal interstitial disease. The percentage of patients in the overall patient cohort with no significant interstitial disease is even higher now, as more recent diagnoses include many people in the early course of the disease. By contrast, lung function loss in asbestos disease from predominately chrysotile exposures is mainly secondary to interstitial disease. In the Libby cohort, interstitial disease generally becomes radiographically visible rather late in the process, and frequently is only a minor factor.

Consistent with the pleural pattern, in the CARD mortality study 43%

(31/72) died with minimal or no interstitial disease (1/0 or less) (Exh. 7 spreadsheets). There was more interstitial disease in the CARD mortality study than in the 123 patients studied in Whitehouse (2004). This is consistent with my clinical observation that interstitial disease often appears in the last years before death.

Consistent with the pleural pattern is the statement in the Peipins et al (2003) abstract: "We observe pleural abnormalities in 17.8% of participants and interstitial abnormalities in less than 1% of participants undergoing chest radiography."

35. Pleural disease in the Libby area is widespread. The patient population is not concentrated in small geographical areas of higher intensity dust contamination from W.R. Grace mining activities. Peipins et al (2003), abstract, states: "Mining, handling, processing and personal or commercial use of asbestos-contaminated vermiculite have led to widespread contamination of the Libby, Montana area." Peipins et al (2003), p.1754, states: "air sampling in downtown Libby in 1975 and at several points in the 1980s detected levels of asbestos well above the OSHA occupational limit of 0.1 fiber/cm3 over eight hours of exposure." A W.R. Grace report, Source Emissions, Results of Surveys, 1975 is attached as Exh. 20. Asbestos fiber levels were stated as 0.67, 1.5 and 1.1 for three locations in the town of Libby in 1975. The town is about seven miles from the mine.